

INTERNAL BROWN SPOT (IBS) -- SOME INTERESTING INSIGHTS

by

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The marketability of Washington potatoes is very dependent on the production of tubers with consistent high quality. The physiological disorder internal brown spot (IBS), first reported in the early 1900's, has become more of a significant problem in the Columbia Basin and has resulted in substantial economic losses in the commercial production of fresh market and processing potatoes. Based on the 1994 production value of the Washington potato crop, a loss of 1% would equate to over four million dollars!

Several different names and terms, including internal rust spot, internal brown fleck, and others, have been used since this disorder was first reported in the literature to identify what appears to be similar internal symptoms. This disorder is characterized by small, irregular, brown or rust-colored necrotic spots or blotches visible inside the vascular ring in the medullary tissue of the potato tuber (1). The necrotic spots can vary in intensity from a rather diffuse and wide blotch area commonly seen in the cultivars Atlantic and Shepody to a more concentrated or localized spot often seen in the cultivars Russet Burbank and Frontier Russet. IBS lesions are scattered throughout the tuber in comparison with brown center and hollow heart which are found in the central core or pith area of the tuber.

Grower and industry experience has indicated that the occurrence and severity of IBS has varied in different years and that certain fields (or growers) may have a higher incidence of the disorder every year. The variation from season to season would indicate that environmental conditions contribute to IBS development. However, differences in production and management practices or definitive climatic factors have not been correlated with an 'IBS year' and 'non- IBS year'. Certain cultivars are more prone to the disorder than other cultivars.

External symptoms of this disorder are rarely seen except in very severe cases. Pathogens have not been associated with IBS, however, several viruses of potato may produce an internal brown necrosis in tubers. IBS is often confused with other internal necrotic symptoms of stem-end discoloration, net necrosis from the leafroll virus, verticillium wilt discoloration, and corky ringspot (often called "spraing" disease) of the tobacco rattle virus.

The developmental stage(s) of the potato plant and tuber, and associated cellular changes, that result in the initiation and manifestation of this disorder have not been clearly identified. There is some evidence in greenhouse studies that IBS can be induced in very small tubers, but in field situations IBS is generally not seen until late in the season or even closer to harvest. However, this should not necessarily infer that initiation was this late in the potato tuber development. The time of initiation of the disorder may be far ahead of the visible manifestation. It is very important to maintain an even and uniform rate of growth, both in the plant and in the tubers, throughout the entire growing season. Any fluctuation in growth rate caused by environmental, water, or pest stress may disrupt the normal cellular development and functioning.

In the growing season of 1995, potato tuber samples were collected on a weekly basis from four different production fields in the Columbia Basin potato area of central Washington State. Tubers were collected from a 4m-length of the same pair of rows for consistency of soil type and other factors during the season. Sampling began when tubers were about 2 cm diameter and continued until harvest. Tubers were weighed and measured individually for size and also specific gravity. One half of the tubers were cut immediately for observation and detection of internal necrosis; the other half of the tubers were placed in storage and cut after a 6-week period. Necrotic areas were fixed in appropriate solutions and different staining procedures were used for the microscopy observations.

Internal brown lesions were evident in very small tubers in all the greenhouse and growth room experiments, but lesions did not become evident in field-grown tubers until mid season. Tuber samples taken from the field and from storage indicated similar expression when observed under the microscope. Our preliminary microscopic examinations have shown that the brown spots were areas of injured and/or localized necrotic cells that had become highly suberized but not necessarily collapsed. The IBS lesions appeared to be areas of dead or dying cells lacking starch grains and with thickened, suberized, and sometimes fragmented cell walls. Both sudan red stain and fluorescence microscopy indicated the presence of suberin around the cell walls. The IBS lesions appeared the same when sampled from different areas of the medullary zone. IBS and brown center lesions appeared to be similar microscopically except that cells in the brown center areas tend to collapse more often. More microscopic examinations must be made to confirm this initial observation. Another observation common in many of the lesion areas was that of patches of tiny dividing cells near the IBS lesions, suggesting an uneven cellular growth rate within the tuber or reaction to the wound signal of the IBS lesion.

Several examples of the microscopic examinations will be presented.

This disorder has been clearly related to a calcium deficiency in various growth room and greenhouse studies by a number of investigators around the world. We reported results to you last year (2) from our controlled environment studies in which IBS lesions were seen as early as tuber initiation in tubers from both +Ca and -Ca fertilization treatments (Table 1). More Ca was present in the peel than the medullary tissue and more Ca was found in the peel under +Ca fertilization. The medullary tissue Ca concentrations did not appear to be greatly influenced by Ca availability in the soil solution, however peel Ca concentrations were significantly affected by Ca availability. Different cells of the potato tuber have also shown differing calcium concentrations (3). It must be emphasized, however, that field studies with various calcium applications have not been as promising and conclusive in the reduction of IBS occurrence.

We have studied various sources and rates of calcium applications on the WSU Othello Research Farm and cooperating grower fields over a ten year period and have found the results to be very inconclusive. Representative data from the 1995 season is shown in Table 2. The lack of a clear relationship of calcium with IBS in field situations certainly illustrates the presence and role of other variables yet to be ascertained. The antagonistic effects of other elements on calcium would be another important factor as well.

In summary, field observations and tuber sampling have provided a large amount of material for observations and microscopic studies. Light, scanning, and transmission electron microscopy methods are being used to observe the development, cellular changes and other characteristics of the disorder internal brown spot in potato tubers. In glasshouse studies, the concentration of calcium in potato tubers was primarily affected by calcium supply. Stress on the tubers caused by high soil temperature increased IBS incidence and severity but only in association with a lack of calcium fertilization. The ultimate realization is that management and careful attention to many details is imperative to avoid these internal disorders as we strive for higher potato yields and plants are pushed closer to their biological maximum.

REFERENCES

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2. Olsen, NL, LK Hiller, LJ Mikitzel, RE Thornton and WL Pan. 1995. Internal brown spot (IBS) development in greenhouse grown 'Russet Burbank' tubers. Proc WA State Potato Conf 34: 29 - 35.
3. Oparka, KJ & HV Davies. 1988. Subcellular localization of calcium in potato tubers. Potato Research 31: 297 - 304.

Table 1. Percent of tubers with IBS as affected by calcium fertility and stage of tuber development [tuber initiation (TI), early bulking (EB), late bulking (LB), and maturity (M)] (see reference 2).

<u>Calcium</u>	<u>Rating</u>	<u>IBS</u> <u>Tuber Stage</u>			
		<u>TI</u>	<u>EB</u>	<u>LB</u>	<u>M</u>
+Ca	mild		24.4	19.4	4.0
	moderate	7.3	7.1	7.4	4.4
	severe		3.3	5.8	3.4
	mild		30.8	12.2	8.4
	-Ca moderate	0.0	3.5	9.7	21.2
	severe		0.0	3.5	11.8
<u>Source of variation</u>		<u>P value</u>	LSD _{0.05}		
Ca			ns		
Tuber stage			ns		
Severity		0.0009	5.1		
Ca*Tuber stage		0.0374	14.1		
Tuber stage*severity		0.0030	14.1		

Table 2. 1995 At-Plant Calcium Trials on Potatoes (summary of two trials in the Quincy and Lind/Othello areas)

Tmt No.	Calcium Treatment	Number of Tubers ^z				Total Defect
		Defect-BC/ Free	HH	IBS	Both	
1	CaSO ₄ 15.6 (Gypsum)	9.6	3.9	0.9	14.4	
2	CaO	17.9	8.9	2.5	0.7	12.1
3	CaCl ₂	16.1	8.3	3.5	1.5	13.9
4	Chelated Ca	15.2	7.8	6.5	0.5	14.8
5	Chelated Ca	18.3	8.0	2.5	1.2	11.7
6	Untreated Ck	16.0	7.3	5.5	1.2	14.0

^z Average of 4 replicates in each trial, 30 tubers cut/replicate